

LECTURE PRESENTATION

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Environmental and genetic factors in age related hearing impairment

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The etiology of age related hearing loss (ARHI), or presbycusis, is multifactorial and includes both genetic and environmental influences. Much research effort has been put into elucidation of the environmental factors and nowadays a huge amount of data are available regarding the effects of noise exposure, ototoxic medication, exposure to chemicals, chronic medical conditions, malnutrition, tobacco smoking and alcohol abuse, although some of these factors remain still controversial [1]. On the other hand, little is known about the role of genetic factors, despite the fact that the number of genetic studies on presbycusis is increasing at a surprising rate in the last few years. The hypothesis that genetic factors could play a significant role in AHRI was proposed as early as the '70s, based on the observation of several families whose members were affected by a presbycusis which manifested very early, occasionally before the age of 45, and the consideration that either the time of onset or the rate of progression of presbycusis vary greatly between individuals. In the '90s, heritability estimates have shown that approximately half of the variance in ARHI is due to heritable factors. An important contribution to the study of genetic factors in ARHI derives from laboratory research on inbred mice. Some of these strains present an early presbycusis and have in common the defective *Cdh23*^{ahl} allele of the gene encoding cadherin23, a protein localized in the stereocilia of the hair cells, which hold the cilia together. Lack or absence of otocadherins increases the vulnerability of hair cells either to aging, or to noise exposure. Experimental data obtained in mice has been found and confirmed in humans. Two genomewide linkage studies and a few association studies based upon candidate genes approaches for AHRI have been published in the last years. AHRI has been correlated with several single

nucleotide polymorphisms (SNPs) in a region of *KCNQ4*; with a SNP in *N-acetyltransferase 2* (confirming the role of oxidative stress in presbycusis); with a highly significant SNP in the *GRHL2* gene; with a significant SNP at the *GRM7* locus (indicating the possible effect of glutamate excitotoxicity in AHRI). These findings are important for both the management and possible future therapeutic intervention into presbycusis.

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Reference

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